Deconstructing Schizophrenia for *DSM-V*: Challenges for Clinical and Research Agendas

Joseph M. Pierre [1]

Abstract

Considerable effort is focused on a revised definition of schizophrenia for the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM)* anticipated in 2012. Among the core debates are how to define a disorder without an established pathophysiology, whether diagnosis is improved by implementing continuous symptomatic dimensions, and to what extent neurocognitive deficits should be integrated into schizophrenia for *DSM-V*. Although lacking in validity, the diagnosis "schizophrenia" remains a useful term for clinical communication, with current antipsychotic therapies encouraging diagnostic "lumping" rather than "splitting." Much evidence supports a move to a dimensional model for psychosis, but doing so raises the potential for overdiagnosis. Validity problems exist not only with schizophrenia, but also with its defining symptoms such as delusions and hallucinations. Integrating dimensional symptom clusters into *DSM-V* schizophrenia, exemplified by models based on neurocognitive deficits, offers a strategic shift of focus onto core symptoms both within, and across, categorical diagnoses. Such a shift is consistent with existing clinical practice, and could pave the way toward more meaningful and scientifically validated reformulations of diagnostic categories in further revisions of *DSM*.

Key Words: Schizophrenia, Psychosis, Diagnosis, *DSM*, Validity

Introduction

With the fifth major revision of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-V)* planned for the year 2012, modifications in the definition and diagnostic methodology of schizophrenia are being thoughtfully considered and debated. Issues pertinent to this debate were the focus of symposia and workshops at the American Psychiatric Association 2007 Annual Meeting (1) and at the 2007 International Congress on Schizophrenia Research (2, 3), and will be further analyzed here. These issues include whether: 1) schizophrenia is a valid disease entity; 2) diagnosis should be based on a dimensional rather than a categorical approach; 3) neurocognition should be incorporated into the diagnostic criteria for schizophrenia; and, 4) the term "schizophrenia" should be discarded altogether.

[1] *UCLA Semel Institute for Neuroscience and West Los Angeles VA Medical Center*

Address for correspondence: Joseph M. Pierre, MD, VA West Los Angeles-Psychiatry, 11301 Wilshire Blvd., Building 210, Room 15, Los Angeles, CA 90073
Phone: 310-478-3711, x43846; Fax: 310-268-4448;
E-mail: drjoe@ucla.edu

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The Validity of Schizophrenia

A fundamental question concerning psychiatric nosology is whether diagnoses should be categorized based on

underlying pathophysiology (causalism) or their clinical manifestations (descriptivism) (4). While the ideal answer would be both, causal models for psychiatric conditions such as schizophrenia remain elusive despite technological advances in genetic screening (5) and neuroimaging (6, 7). As a result, *DSM-IV* (8) is filled with descriptive diagnoses, called "disorders," that are really "syndromes" rather than "diseases."

The failure to uncover a unitary pathophysiology has led to the general belief that, in keeping with Bleuler's original conceptualization, "schizophrenia" is a descriptive term that encompasses a variety of different disease processes. If this is so, and schizophrenia is not a "disease entity" *per se*, but rather a collection of overtly similar but biologically heterogeneous conditions, then the establishment of diagnostic criteria for "schizophrenia" in *DSM-III* (9) and *DSM-IV* may have greatly improved upon clinician interrater reliability while doing little to validate schizophrenia as a disease concept (10). Without established etiologic validity for schizophrenia, some have argued that the term and/or concept be abandoned in *DSM-V* (2).

The term "schizophrenia" provides clinicians with a meaningful descriptor of a commonly appearing constellation of observable human behaviors associated with poor functioning (i.e., a "syndrome"), despite the fact that there may be significant underlying pathophysiologic heterogeneity.

Medical diagnosis serves different functions, including communication with others ("communication diagnosis"), identification of a group for therapeutic intervention ("treatment diagnosis"), and definition of an underlying pathophysiology ("etiological diagnosis") (11). With communication diagnosis, a Kraepelinian lumping strategy, even in the absence of causal validity, can still be practical. Take clouds, for example. Having inherent face validity, few spend time debating whether a cloud is a cloud. But while a cloud can be defined as a visible aggregation of water vapor in the sky, there are a wide variety of cloud subtypes (e.g., cumulus, stratus, cirrus, etc.), each named based on their external appearance, location and the conditions that give rise to them. Some clouds might be better characterized as mist or fog, while still others might look like a cloud, but upon closer inspection, might be better described as a contrail, smog or even a swarm of bees. Yet despite the underlying variability within the term, "cloud" still serves a practical purpose in everyday communication. Such is the state of schizophrenia. The term "schizophrenia" provides clinicians with a meaningful descriptor of a commonly appearing constellation of observable human behaviors associated with poor functioning (i.e., a "syndrome"), despite the fact that there may be significant underlying pathophysiologic heterogeneity.

The clinical utility of a lumping strategy for schizophrenia is strengthened when Bleulerian splitting does little to enhance treatment diagnosis. At present, existing *DSM-IV* schizophrenia subtypes have almost no relevance in terms of selecting a more effective therapy. Given this state of affairs, retaining the broad descriptive diagnosis "schizophrenia" in *DSM-V* is justified from a communication and treatment perspective (i.e., "clinical utility"), even in the absence, for now, of biologic or etiologic validity (12, 13).

In medicine, therapeutic discoveries, both deliberate and accidental, have a profound impact on all aspects of diagnosis—communication, treatment and etiologic. The state of therapy, therefore, directly impacts the need for diagnostic revision as governed by clinical utility. Currently, antipsychotic pharmacotherapy is a fairly homogeneous armamentarium that is prescribed (and FDA approved) for an ever-increasing number of conditions, from schizophrenia and bipolar disorder to unipolar depression and anxiety disorders. If antipsychotic medications were the only available therapies, and if all patients responded, one could empirically argue that differentiating schizophrenia from these other conditions is unwarranted. But, of course, not all patients do respond, therapies often do have symptomatic and diagnostic specificity, and there are several different aspects of treatment, including remediation of symptoms, relapse prevention, functional rehabilitation and the specific targeting of etiologic lesions. This complexity highlights a limitation of clinical utility, in that the deliberate development of therapies that are curative rather than merely palliative necessitates diagnostic splitting, at least in the research laboratory.

With the advent of *DSM-III*, diagnostic categories were revised to be deliberately "atheoretical," or disassociated from any attributed psychological or pathophysiologic cause since, for most disorders, etiology was disputed or unknown. As such, the intent of *DSM-III* was to provide a "descriptive" definition of different types of "mental disorders," loosely defined as a "clinically significant behavioral or psychological syndrome or pattern that occurs in an individual" that is associated with disability or distress (9). At the same time, both *DSM-III* and *DSM-IV* purport to "provide clear descriptions of diagnostic categories in order to enable clinicians and investigators to diagnose, communicate about, study, and treat people with various mental disorders" (8, 9). Although the hope was that pathophysiology would eventually be determined, the lack of etiologic validity for disorders in the twenty-eight years since *DSM-III* suggests that it may

be necessary to critically reevaluate whether *DSM* can satisfy both clinical and research agendas. If the view that schizophrenia represents a multitude of underlying conditions is correct, then it would be best to steer away from the use of descriptive communication diagnosis in research, particularly when searching for etiology, since most human studies that attempt to "find a lesion" are conducted with subjects recruited by *DSM* diagnosis and, therefore, doomed from the start (11).

Categories and Dimensions

A commonly proposed alternative to categorical diagnosis involves the modeling of disorders in terms of "spectrum" or "dimensional" illness (14). In the simplest sense, the term "dimensional" is invoked to describe a quantifiable trait that spans a continuum from normal to pathologic. Proponents of a dimensional approach for schizophrenia argue that psychosis is such a trait, and not simply a "yes" or "no" phenomenon. A substantial body of research now supports the notion that psychosis is distributed on a dimensional continuum that extends into the "normal" population. Epidemiologic studies have consistently demonstrated the presence of "subthreshold" psychotic symptoms in general nonhelp-seeking populations (15, 16). These detected symptoms are typically found, on closer examination, to be nondistressing and, therefore, of limited clinical significance; but, the findings, nevertheless, indicate that psychotic features are distributed along the continuum from pathologic to normal (14, 17, 18).

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However, if dimensional or spectrum models are reality and categorical borders are illusory, this presents another assault on diagnostic validity. For example, the reality of clouds is that while they are aggregations of water vapor with observable borders, there is less densely collected, unseen water vapor in the air surrounding clouds. While a visible border seems apparent, at a physical level a defined border between "cloud" and "not-cloud" is much harder to delineate, and if no "real" boundary exists, then the categorical entity "cloud" can hardly be regarded as a valid entity. That same problem arises with schizophrenia. While seemingly contradictory, this duality between category and continuum is present throughout medicine, whether in hypertension (where there is now "prehypertension"), cancer (where there is carcinoma *in situ*), and even debates about what constitutes life and death. In fact, while human perception is particularly adept at contriving patterns and boundaries in "things," the reality is that most "things" can be conceptualized along both categorical and dimensional terms, as with the "particle" and "wave" duality of light.

In this spirit, it appears likely that *DSM-V* will incorporate dimensional aspects within categorical diagnoses (14, 19). In fact, this was considered for *DSM-IV* (in which it is stated, "there is no assumption that each category of mental disorder is a completely discrete entity with absolute boundaries dividing it from other mental disorders or from no mental disorder"), but was ultimately abandoned due to "serious limitations" (8). One such limitation is that while a continuous view of disease states may more closely approximate reality, categorical distinctions are vital to clinical decision making. Therefore, even if schizophrenia is conceptualized as a spectrum illness, clinicians must still draw a "line in the sand" and define its categorical presence or absence, so that they can decide when to initiate treatment (and so that insurance companies can decide when to reimburse).

DSM has historically defined that line based on the presence of "significant" impairment and/or distress, but such variables can be highly subjective and value embedded. For example, delusions and hallucinations may or may not be subjectively distressing, and poor insight or grandiosity may even play a protective role among some psychotic individuals (20). But psychotic symptoms that are not subjectively distressing (e.g., "ego-syntonic") often do lead to social dysfunction as a result of their effect on, and the reaction of, others. For example, stigmatization has been shown to have a negative impact on the course of illness in schizophrenia and is predictive of distress (21). In this way, unlike medical conditions in which morbidity is typically defined by the hastening of death, psychiatric morbidity and pathology can sometimes be defined not only by an individual's symptoms themselves but by society, or the dynamic interaction between patient and culture. Future research addressing the psychotic spectrum must seek to elucidate whether the continuum between normal and pathological is linear (22) and to determine the relevant factors, both individual and cultural, that convert subthreshold traits into threshold symptoms (e.g., the mediators of distress and functional impairment) (23).

Research seeking to predict the onset of schizophrenia before it occurs, by characterizing an attenuated "prodromal" form of the illness, illustrates some of the challenging issues surrounding expansion of the psychotic spectrum. Prodromal research criteria are largely based on "subthreshold" psychotic symptoms, though neurocognitive, neuroimaging, psychophysiologic and genetic markers of psychotic risk are also being sought with the hope that primary or secondary prevention might eventually be possible. Research

centers identifying help-seeking subjects meeting entry criteria for prodromal psychosis have published "conversion rates" (e.g., progression to clear psychosis) of 21 to 54% at 1 year (24). Recent pooled data from North American centers indicate a conversion rate of 35% at 2.5 years (25). These rates are substantially greater than the background rate of psychosis in the general population, suggesting that the prodrome may represent a valid place in the psychotic spectrum and a predictor of eventual progression to full-blown psychosis or schizophrenia. But confounding these research efforts is the fact that therapeutic interventions are, by necessity, being implemented and investigated in these studies at the same time, with competing goals. For example, without more controlled trials (26, 27), it is difficult to interpret conversion rates—does a 40% conversion rate suggest that 60% of subjects were mislabeled as prodromal or that the intervention was effective in preventing conversion? Also, a substantial proportion of prodromal patients are referred to research programs already on antipsychotic medication (25, 28), though it is not yet clear whether antipsychotics are efficacious or safe in this population, or whether other interventions such as antidepressants or omega-3 fatty acid supplementation might suffice (29, 30). Clinicians justify assertive pharmacotherapy in the absence of a *DSM-IV* diagnosis based on patient's distress and a desire for help, but also on untested assumptions about efficacy and a spectrum view of psychosis. Without established disease validity and evidence-based treatments, integrating a spectrum model of psychosis into *DSM-IV* that recognizes subthreshold symptoms encourages and legitimizes such practice, though it may not be in the best interest of patients.

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While the antipsychiatrists warn that diagnosis can serve as a tool of social control (e.g., "political delusions" in China), in our current era and society, drawing the line of pathology at anywhere other than "perfect" functioning is an equally foreboding risk. The potential for expansion of pathologic labels into normal variation, and the acknowledgment that such efforts could benefit those with vested interests in psychiatry as an industry, has led some to rally against "disease mongering" (31). After all, if human traits are biologically determined and distributed along continua, one could argue that anything less than ideal height, attractiveness, intelligence, cognitive function, social ease, anxiety or psychoticism could impair functioning and, therefore, be a potential target for a diagnostic label and intervention. Shyness, baldness, shortness, loneliness, flat feet, road rage, diminutive sex organs—no doubt each has some biological basis and could detract from ideal functioning, but is that enough to make a disorder or disease (31, 32)? Such issues are core to debates about diagnostic revision in *DSM-V*, where the proposed dimensionalization of schizophrenia opens the door to a potential for overdiagnosis, as it has with other conditions that share symptomatic and possible etiologic overlap with it, such as bipolar disorder (33, 34) or autism (35, 36). The intrusion of diagnostic labels onto traits and behaviors previously considered normal threatens the legitimacy of psychiatry as a medical field. At its worst, it paves a path from well-intentioned cosmetic psychopharmacology to the perils of eugenics. With the introduction of continuous dimensional constructs for schizophrenia, categorical distinctions between healthy versus disorder, trait versus symptom, and cosmetic versus therapeutic intervention need to be considered most carefully.

Symptomatic Criteria

Although the category-continuum duality of schizophrenia is an important issue driving diagnostic revision for *DSM-V*, critical thinking about the symptoms that represent diagnostic criteria has largely been pushed aside in American psychiatry. This neglect is striking given that many of the same difficulties stemming from the category-continuum duality of schizophrenia arise during the clinical assessment of its proposed symptoms. For example, delusions are defined in the glossary of *DSM-IV* as categorical all-or-nothing phenomena, but in practice it can be quite challenging to identify when a delusion is present (37, 38). No firm guidelines are established in *DSM-IV* to reliably distinguish delusions from "fanatical" political beliefs or religious faiths (39) or, for that matter, from "overvalued ideas" (40) or some "normal" beliefs (41). The term "bizarre," vital to the differentiation of delusions in delusional disorder as opposed to schizophrenia, has been found to have low rates of interrater reliability, leading many to call for elimination of the concept (42-45).

Similar diagnostic dilemmas arise when considering hallucinations. Given clinical variations in localization, voice quality, insight and associated distress, it seems likely that not all patients who "hear voices" are experiencing the same physiologic process. If that is so, then the term "hallucination," like schizophrenia, may be a broad label for a variety of distinct experiences. Perhaps this is why auditory verbal hallucinations (i.e., "hearing voices") are ubiquitous

among psychiatric illnesses, including not only psychotic, mood, substance abuse and cognitive disorders, but those in which little or no mention of hallucinations is made in *DSM-IV*, such as conversion disorder (46, 47), borderline personality disorder (48), sexual abuse (49, 50), grief (51), and posttraumatic stress disorder (52, 53). Patients who endorse "voice hearing" in the absence of any other psychiatric symptoms (i.e., "monosymptomatic hallucinations") are also common in community samples and clinical practice (15-18, 22). Attempts to resolve this apparent contradiction have resulted in the terms "pseudohallucinations" (for less than hallucinatory experiences) or "nonpsychotic hallucinations" (for hallucinations in otherwise nonpsychotic individuals), but neither concept has a consistent definition or validity (54, 55). Cognitive theories of auditory verbal hallucinations often invoke the misattribution of "inner speech" to an external source (56). That view fails to fully account for a number of clinical features of voice hearing (e.g., multiple voices, voices that sound like someone else, voices that are clearly distinguished from usual inner speech, etc.), but if correct, even in some cases, it then begs the question of how an auditory verbal hallucination is distinct from a deficit in insight, a delusion, thought insertion, a misidentification experience (i.e., "alien-voice syndrome"), or whether it is an "hallucination" at all. Resolving these many questions about symptoms is of paramount importance to the establishment of valid disease diagnosis. It is likely, for example, that the presumed heterogeneity of schizophrenia and the variety of individual treatment responses may be due, at least in part, to improper characterization of core symptoms.

If schizophrenia is to be properly deconstructed, its building blocks, including positive symptoms, must be carefully inspected so as not to build a house of cards.

If schizophrenia is to be properly deconstructed, its building blocks, including positive symptoms, must be carefully inspected so as not to build a house of cards. Unfortunately, interest in the phenomenology and validity of positive symptoms has been largely absent from American psychiatric research, leading Andreasen to call for a renaissance of clinical researchers dedicated to descriptive psychopathology (57, 58). An inadvertent side effect of the descriptive approach taken in *DSM-III* and *DSM-IV* has been a shift away from an understanding of internal experiences to, in many cases, a cursory checklist approach to patient assessment. This diagnostic approach precludes the discernment of fine distinctions of subjective experience and, instead, encourages symptomatic lumping.

In Europe, Bentall has argued for the abandonment of the categorical diagnosis of schizophrenia altogether (along with other *DSM-IV* conditions) in favor of a research and clinical focus on patients' symptoms (or more properly "complaints," since the term "symptom" implies a disease) (59). While such proposals have merit, *DSM* focuses on "syndromes" and "disorders" because symptoms do appear to cluster together in stereotypic patterns, suggesting some common underlying pathophysiology. The integration of symptom dimensions into categorical diagnoses in *DSM-V* may offer a happy medium in this regard. For one, dimensionalization of symptoms could lead to a more careful scrutiny of symptoms themselves, both in terms of clinical phenomenology (e.g., examining more closely what differentiates a symptom and a normal trait) and symptom-focused etiologic research (e.g., exploring the pathophysiology of symptoms as opposed to disorders). For this to occur, dimensional quantification will require more detail than just a Likert scale of severity ratings, but guidelines on how to assess features of symptoms that govern severity, distress and pathology. Such a shift, especially if the same dimensions are included across diagnostic categories whenever possible, also might facilitate a decreased reliance on existing categorical boundaries in favor of increased recognition of dimensional commonalities. That, in turn, could help to establish firmer etiologic links between disorders. Discoveries arising from such a paradigm shift will be vital to the eventual reorganization and reformulation of more valid diagnostic boundaries in further *DSM* revisions.

Neurocognitive Deficits in Schizophrenia

The "deconstruction" movement in schizophrenia seeks to disassemble the existing categorical diagnosis into better-defined working parts, integrating data from genetics, neuroimaging, psychology and other disciplines, and then group symptoms that cluster together in order to rebuild them into a more valid working definition of schizophrenia. This is another, more complicated way in which the term "dimensionalization" is used—to describe the reorganization of co-occurring symptoms, based on factor analytic studies, into clusters or dimensions that can be quantified together along a continuum. It has long been proposed that schizophrenia be subdivided into dimensions such as positive, negative and disorganized (60, 62), while more recent proposals add mania and depression for a five-factor model (14, 19). By allowing each symptom dimension to be quantified, such models have been heralded as a way to differentiate between categorical diagnoses, such as schizophrenia and bipolar disorder, where there is significant symptomatic overlap (14, 19, 61, 62). Likewise, the creation of validated clustered dimensions such as negative symptoms or cogni-

tive deficits encourages both a clinical and research focus on aspects of schizophrenia that have been historically neglected, are functionally pertinent, and may represent symptoms that have different etiologies that might, therefore, respond to different types of intervention. It is in this fashion that category and continuum may be best integrated in *DSM-V*.

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Cognition in schizophrenia offers a good example of how working from isolated symptom, to clustered symptom dimension, to categorical diagnosis can contribute to testable models for disorders that aren't just syndromes based on gross pattern recognition. Although cognitive impairments are the hallmark of "cognitive disorders" such as dementia, they are also found in a variety of other *DSM* syndromes including schizophrenia. The term "cognition" is a broad category used by both clinicians and researchers and is often defined by enumerating its subcategories (e.g., memory, attention, language, etc.), all of which are conceptualized as continuous dimensional traits and quantifiable by tests designed to elicit specific deficits. Thus, different neurocognitive abilities are distinguishable from one another and are conceptualizable as dimensional constructs with categorical cut-offs that demarcate "normal" and "deficit." Cognitive impairments in schizophrenia are increasingly recognized as core, functionally pertinent deficits that should, therefore, be included in *DSM-V* (63, 64). A consensus neurocognitive battery for schizophrenia has been developed and is already being implemented in clinical trials (65).

There is substantial evidence that characteristic neurocognitive deficits are present long before a patient meets *DSM-IV* criteria for schizophrenia, and that the cognitive dysfunction has both genetic and structural correlates (66). Similarly, specific cognitive deficits are found not only in patients with *DSM-IV* schizophrenia, but in schizophrenia-spectrum conditions and in relatives of patients with schizophrenia, suggesting a genetic basis, or "endophenotype" (67) (an endophenotype is a measurable, heritable and state-independent trait that is associated with illness, cosegregates with that illness in families, and is found at a greater rate among unaffected family members compared to the general population) (68, 69). Several investigators have extended this line of research to propose hypothetical disease categories based on core neurocognitive impairments, such as "schizotaxia" (70, 71), "cognitive dysmetria" (72, 73), or the shared endophenotypic features of schizophrenia and schizotypy (74). For example, Andreasen posited that "schizophrenia is a single illness with a single phenotype … defined by a fundamental cognitive abnormality" and that this abnormality ("cognitive dysmetria") gives rise to second-order neurocognitive dysfunction, as well as gross psychotic symptoms such as delusions or disorganization (73). While this and other models require validation, they are at least based on fundamental deficits that have greater validity than existing symptomatic criteria for schizophrenia. Such "bottom-up" research efforts are more likely to succeed in developing validated disease models than "top-down" strategies that work backwards from unvalidated categorical diagnoses.

A solid case, therefore, can be made to incorporate cognitive deficits into diagnostic criteria for schizophrenia in *DSM-V*. The biggest challenge, then, is how to include neurocognitive criteria that can be readily elicited by clinicians and that are well-correlated with observable functional improvements. For the most part, the cognitive deficits characteristic of patients with schizophrenia are invisible to the untrained clinician's eye. Existing cognitive batteries are too cumbersome to be incorporated into routine clinical work, and ratable deficits do not necessarily mirror clinical behaviors, limiting their relevance for clinicians (75). To integrate cognitive deficits into *DSM-V*, schizophrenia could follow the example of dementia by incorporating symptom dimensions (e.g., memory, attention, social cognition, etc.) that may require more detailed or specialized clinical assessment (64, 65). Just as clinicians are able to complete bedside screening of cognitive impairment in dementia, current efforts to develop mini cognitive assessments for schizophrenia (76-79) could facilitate the clinical assessment of core neurocognitive deficits in schizophrenia.

DSM-V and Beyond

Many of the points discussed here were considered thoroughly following the publication of *DSM-III* and in anticipation of *DSM-IV* (10, 80-83), but remain unresolved with the coming of *DSM-V*. Much of the difficulty in deconstructing schizophrenia for this next revision involves an ongoing tension between conflicting approaches (e.g., "lumping" versus "splitting," "top-down" versus "bottom-up") that are sometimes better suited to different research or clinical aims. Recall our definition of "cloud" as a *visible* aggregate of water vapor—in most everyday situations that definition suffices. But for nephologists hoping to determine the essence of clouds, what causes them, and their meaning

in a larger context, more rigorous distinctions between *altocumulus castellanus* and *altocumulus lenticularis* clouds are useful. In this way, *DSM* should retain its role as a "rough guide" for clinical work and focus on that which is visible to clinicians. That means retaining the categorical diagnosis of schizophrenia for now, even though doing so has contributed to stagnation in etiologic research and novel therapeutic development. At the same time, incorporating symptom dimensions, including cognitive deficits within schizophrenia for *DSM-V* will sanction operation outside the confines of categorical boundaries in order to further our understanding of what schizophrenia really is, to determine its essential components, and to discover what makes it apparent and relevant to clinicians and patients alike. Once those goals are achieved, and not before, a more radical restructuring of *DSM* diagnostic categories will be in order.

P.S. What's in a Name?

Finally, given aforementioned problems with validity, as well as considerable stigmatization from the label, some have proposed that we eliminate the term "schizophrenia" from *DSM-V* altogether (1, 84-86). As discussed above, the validity issue goes well beyond semantics and will not be remedied by a name change. With regard to stigmatization, certainly the abandonment of "inadequate personality disorder" was well-advised, but changing names for essentially the same condition, as from "multiple personality" to "dissociative identity disorder" or "mental retardation" to "pervasive developmental disorder," seems more of a parlor trick. The stigma associated with schizophrenia arises mainly because of our inability to treat it effectively, rather than its name. That needs to change. As we inch closer to valid disease models and curative treatments, perhaps the stigmatization associated with mental illness in general and, in turn, the terms "psychiatry" and "psychiatrist" can be reduced as well.

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